Journal of Allergy and Clinical Immunology
Volume 110 • Number 2 • August 2002

Housing characteristics, reported mold exposure, and asthma in the European Community Respiratory Health Survey

Jan-Paul Zock PhD[a]
Deborah Jarvis MD[b]
Christina Luczynska PhD[b]
Jordi Sunyer MD[a]
Peter Burney MD[b]

on behalf of the European Community Respiratory Health Survey*

Key words

Asthma

housing

dampness

mold

carpeting

European Community Respiratory Health Survey

[a] the Respiratory and Environmental Health Research Unit, IMIM, Barcelona, and [b] the Department of Public Health Sciences, King's College, London.

*Principal participants of the European Community Respiratory Health Survey are listed in the Appendix.

The coordination of this work was supported by the European Commission. The following grants helped to fund the local studies: Australia: Allen and Hanbury's, Australia; Belgium: Belgian Science Policy Office, National Fund for Scientific Research; Estonia: The Estonian Scientific Foundation (grant 1088), Glaxo Welcome; France: Ministere de la Santé, Glaxo France, Institut Pneumologique d'Aquitaine, Contrat de Plan Etat-Région Languedoc- Rousillon, CNMATS, CNMRT

(90MR/10, 91AF/6), Ministre delegué de la santé, RNSP; Germany: GSF and the Bundesminister für Forschung und Technologie, Bonn; India: Bombay Hospital Trust; Italy: Ministero dell'Università e della Ricerca Scientifica e Tecnologica, CNR, Regione Veneto grant RSF no. 381/05.93; The Netherlands: Ministry of Welfare, Public Health and Culture; New Zealand: Asthma Foundation of New Zealand, Lotteries Grant Board, Health Research Council of New Zealand; Norway: Norwegian Research Council project no. 101422/310; Spain: Ministero Sanidad y Consumo FIS grants no. 91/0016060/00E-05E, no. 92/0319, no. 93/0393, Hospital General de Albacete, Hospital General Juan Ramón Jiménez, Consejeria de Sanidad Principado de Asturias; Sweden: The Swedish Heart Lung Foundation, the Swedish Medical Research Council, the Swedish Association against Asthma and Allergy; Switzerland: Swiss National Science Foundation grant 4026-28099; United Kingdom: National Asthma Campaign, British Lung Foundation, Department of Health, South Thames Regional Health Authority; United States: United States Department of Health, Education and Welfare Public Health Service Grant no. 2 S07 RR05521-28.
Received for publication December 26, 2001.
Revised May 6, 2002.
Accepted for publication May 10, 2002.

Reprint requests: Jan-Paul Zock, PhD, Municipal Institute of Medical Research (IMIM), Dr Aiguader 80, E-08003 Barcelona, Spain.

0091-6749/2002 $35.00 + 0 **1/81/126383**

Barcelona, Spain, and London, United Kingdom

Background: The effects of home dampness and mold exposure on adult asthma are not clear.
Objective: We aimed to investigate the associations between housing characteristics related to dampness, mold exposure, and house dust mite levels and adult asthma in 38 study centers from the European Community Respiratory Health Survey.
Methods: Data about the present home, heating and ventilation systems, double glazing, floor covers, recent water damage, and mold exposure were obtained by means of an interviewer-led questionnaire. The associations between these factors and asthma, as defined on the basis of symptoms in the last year, and of bronchial responsiveness, as determined with methacholine challenge, were evaluated. Odds ratios (ORs) were obtained by using random-effects meta-analyses adjusted within study centers for sex, age group, and smoking status.
Results: Fitted carpets and rugs in the bedroom were related to fewer asthma symptoms and bronchial responsiveness (OR range, 0.69-0.91). This effect was consistent across centers and more pronounced among house dust mite-sensitized individuals. Reported mold exposure in the last year was associated with asthma symptoms and bronchial responsiveness (OR range, 1.14-1.44). This effect was homogeneous among centers and stronger in subjects sensitized to *Cladosporium* species. In centers with a higher prevalence of asthma, the prevalence of reported indoor mold exposure was also high. This association was observed for reported mold exposure by asthmatic subjects (Spearman $r_s = 0.46$), as well as reported mold exposure by nonasthmatic subjects ($r_s = 0.54$). Reported mold exposure was highest in older houses with recent water damage.
Conclusion: We conclude that indoor mold growth has an adverse effect on adult asthma.

Abbreviations used

ECRHS
European Community Respiratory Health Survey
OR
Odds ratio

A number of studies in different countries have shown adverse effects of dampness and mold exposure in homes on adult

respiratory health, particularly for symptoms of wheezing and cough.[1] [7] The effect of dampness and mold exposure on adult asthma is less clear, and the association with bronchial responsiveness has not been studied to date. Published data consist of separate studies with different instruments to assess exposure to dampness and mold (eg, history of water damage, visible damp stains, visible mold spots, odors of mold, and perception of damp and mold problems). Different climatic conditions and housing characteristics between countries are likely to cause important differences in exposure to dampness and molds. Thus there is a need for information from an international study on adult asthma using a common protocol for assessment of exposures to dampness and mold and asthma outcomes, including bronchial responsiveness.

The European Community Respiratory Health Survey (ECRHS), an international multicenter epidemiologic study on asthma, collected standardized information on asthma prevalence and known or suspected risk factors for atopy and asthma.[8] To date, single-center analyses in Sweden,[9] [10] Germany,[11] and Australia[12] have shown relationships between damp housing, levels of viable molds, and house dust mite allergens and asthma symptoms, but no single publication has comprehensively described the association between home dampness, mold exposure, and asthma across all participating centers.

The objective of the present analysis was to investigate the associations between housing characteristics related to dampness, mold exposure, and house dust mites levels and adult asthma in all areas of the ECRHS.

Methods

Study population and questionnaire

The methodology for the ECRHS has been described elsewhere.[8] In this analysis data of a random general population sample from 38 study centers were included.[13] Centers were located both in Europe (Belgium, Denmark, Estonia, France, Germany, Iceland, Ireland, Italy, The Netherlands, Norway, Spain, Sweden, Switzerland, and United Kingdom) and outside Europe (Australia, India, New Zealand, and the United States).

An interviewer-led questionnaire collected information on respiratory symptoms, self-reported asthma and allergic disorders, and environmental and lifestyle factors. Participants were asked whether they had experienced the following symptoms in the last 12 months: (1) wheezing with breathlessness; (2) wheezing apart from colds; (3) being woken by an attack of shortness of breath; (4) asthma attack; and (5) use of current medication for asthma. Current asthma was defined as the presence of at least one of factors 3, 4, or 5 listed above.[8]

The information on housing characteristics obtained in the interview included age and type of the present home, type of heating and ventilation systems, presence of double glazing, type of floor cover in the bedroom and living room (the latter defined as the room subjects used most at home during the day), any water damage to the house (broken pipes, leaks, or floods) in the last 12 months, presence of water collecting on the basement floor in the last 12 months, and mold or mildew on any surface inside the home in the last 12 months.

Bronchial reactivity and allergy testing

FEV_1 was recorded by means of spirometry by using a stand-ardized method.[14] Methacholine challenge was carried out with a dosimeter (Mefar, Brescia, Italy). Bronchial responsiveness was defined as a fall of at least 20% in FEV associated with a methacholine dose of 1 mg or less.

Specific serum IgE levels against the mold *Cladosporium herbarum* and against the house dust mite *Dermatophagoides pteronyssinus* were determined by using the Pharmacia CAP System (Pharmacia Diagnostics, Uppsala, Sweden). Sensitization was defined as a specific IgE level of greater than 0.35 kU/L. Skin prick testing against the molds *Alternaria alternata* and *C herbarum* was carried out by using Phazets (Pharmacia Diagnostics). Sensitization was defined as a wheal diameter of larger than 0 mm.

Analysis

Questionnaire data were available for 19,218 subjects from 38 centers. Excluded from statistical analyses were 164 subjects with missing data for smoking habits and an additional 181 subjects with missing data for one of the 5 symptom questions mentioned earlier.

Results of bronchial responsiveness testing, blood IgE analyses, and skin prick testing were available for 13,340 (74%) subjects, 13,915 (78%) subjects, and 15,202 (85%) subjects, respectively, each from 36 centers. There were no major differences in respiratory health outcomes between responders and nonresponders to these tests. The only apparent difference was that fewer women (68%) than men (80%) underwent valid bronchial responsiveness testing basically because of exclusion of pregnant women and of subjects with a baseline FEV_1 of either less than 1.5 L or less than 70% of the predicted value.

Associations between housing characteristics and dichotomous health outcomes were evaluated by using prevalence odds ratios (ORs) obtained in unconditional logistic regression analyses. ORs and SEs adjusted for sex, age group, and smoking status were estimated separately for each study center. Average effect estimates were derived, and potential heterogeneity between centers was examined by using standard methods for random-effects meta-analysis.[15] In some of the meta-analyses, a number of centers had to be dropped from the analysis because of insufficient data. Heterogeneity between center-specific risk estimates was considered significant if the associated *P* value was less than .1. Potential modification of an association by a third factor was evaluated by calculating the *P* value for multiplicative interaction in logistic regression models, with a fixed adjustment for either center or country.

Results

There was a large variation in the prevalence of asthma symptoms and bronchial responsiveness across the study centers (Table I).

Table I. General and respiratory health characteristics of a random population sample from 38 study centers in 18 countries (N = 18,873)

	No.	%	Minimum and maximum across centers (%)
Men	8967	47.5	40-54
Women	9906	52.5	46-60
Age group, 20-29 y	6462	34.2	18-49
Age group, 30-39 y	7705	40.8	34-51
Age group, 40-45 y	4706	24.9	14-38
Never smoked	8360	44.3	30-86
Former smoker	3792	20.1	3-27
Current smoker	6721	35.6	10-57
Wheezing and breathlessness in the last year	2129	11.3	1-22
Wheezing apart from colds in the last year	2509	13.3	3-25
Current asthma*	1613	8.5	3-16
Bronchial responsiveness†	1913	14.3	4-29
Specific IgE to house dust mite‡	2819	20.3	7-35
Specific IgE to *C herbarum*§	525	3.8	0.3-13
Positive skin prick test response to *C herbarum*‖	362	2.4	0.0-11
Positive skin prick test response to *A alternata*¶	712	4.7	0.3-15

*†‡§‖¶

*Asthma symptoms, medication use, or both in the last 12 months.
†Methacholine provocation test (methacholine dose of 1 mg or less causing a fall of 20% in FEV_1); data available for 36 centers (n = 13,340).
‡Specific IgE titer (CAP) to *D pteronyssinus* of greater than 0.35 U/mL; data available for 36 centers (n = 13,915).
§Specific IgE titer (CAP) to *C herbarum* of greater than 0.35 U/mL; data available for 36 centers (n = 13,915).
‖Skin prick test reaction to *C herbarum* of greater than 0 mm; data available for 36 centers (n = 15,200).
¶Skin prick test reaction to *A alternata* of greater than 0 mm; data available for 36 centers (n = 15,202).

The highest prevalence rates were observed in English-speaking countries. One fifth of the study population was sensitized to house dust mite allergen, with the highest rates in Ireland, the Netherlands, New Zealand, and Australia. Overall *C herbarum* sensitization was less prevalent but was higher than 10% in the Netherlands. *A alternata* sensitization was common in Australia and the United States. In total, 895 (5.9%) subjects had positive skin test responses to either *A alternata* or *C herbarum*. Housing characteristics varied substantially among the centers (Table II).

Table II. Associations between current housing characteristics and asthma outcomes (meta-analyses)

	Overall (%)	Range (%)	Wheezing and breathlessness in last year	Wheezing apart from colds in last year	Current asthma	Bronchial responsiveness

ORs (95% CIs) adjusted within study centers for sex, age group, and smoking status are shown where indicated.
*$P < .10$, test for heterogeneity.
†Reference group had neither fitted carpets nor rugs in living room.
‡Reference group had neither fitted carpets nor rugs in bedroom.
§Reference group had either no basement or dry basement.

Central heating was negatively associated with wheezing but not with asthma or bronchial responsiveness (Table II). The presence of ducted air heating was positively associated with asthma. Air conditioning was associated with an increased prevalence of wheezing with breathlessness and of current asthma. The estimates from the meta-analyses were based on data from 19 and 14 centers, respectively. These associations remained statistically significant after adjustment for the other housing characteristics in Table II (results not shown). The presence of rugs in the living room was negatively associated with wheezing and asthma. Having fitted carpets or rugs in the bedroom was related to a lower prevalence of symptoms and bronchial responsiveness. These associations did not change to a large extent after adjusting for the other housing characteristics under study. Table II further shows that water damage and the presence of mold in the home were related to asthma symptoms, and mold exposure was also related to bronchial responsiveness. In models with mutual adjustment, ORs for mold exposure remained statistically significant, whereas the ORs associated with water damage reduced toward unity and lost statistical significance (results not shown). Adjustment for level of education did not alter the risk estimates presented in Table II.

The effect of fitted carpets and rugs in the bedroom on asthma was investigated in more detail. Because the relationships between fitted carpets and current asthma were similar to those of rugs and asthma (Table II), these 2 groups were joined to evaluate the association between any textile floor cover in the bedroom and current asthma by study center (Fig 1).

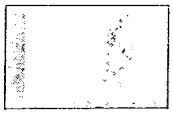

Fig. 1. Carpets, rugs, or both in the bedroom and current asthma by study center (ranked from low to high according to the frequency of carpets, rugs, or both in the bedroom). ORs and 95% CIs, adjusted within study centers for sex, age group, and smoking status, are shown. The size of each *square* is proportional to the reciprocal of the variance of the estimate for the center. The *diamond* indicates 95% CI of the combined OR from the model, with study center as the random effect. Thirty-four centers could be included ($P = .54$, test for heterogeneity).

Results from 4 centers had to be excluded in the meta-analysis because of limited numbers of unexposed asthmatic patients. Fig 1 shows a homogeneous negative association between the presence of carpets, rugs, or both in the bedroom and current asthma, without evidence for different associations in centers in which bedroom carpeting was more or less common.

Analyses were done after stratification for specific house dust mite sensitization to evaluate the possible modifying effect of house dust mite allergy on the relationship between bedroom carpeting and asthma. ORs for all health outcomes were lower among individuals *with IgE* sensitization to house dust mite (results not shown). This difference was statistically significant (P for interaction = .05) for bronchial responsiveness, with the protective effect of having any textile floor cover being stronger among mite-sensitized subjects (OR, 0.74; 95% CI, 0.57-0.97) than among nonsensitized individuals (OR, 0.90; 95% CI, 0.75-1.09).

The association between reported mold exposure in the last year and current asthma, as observed in Table II, was studied in more detail. First, to investigate possible overreporting or underreporting of mold exposure related to asthma status, we evaluated the ecological relationship between community prevalence of asthma and the percentage of subjects reporting mold exposure. There was a significant ecological association between asthma and mold exposure (Spearman $r_s = 0.55$, $P < .001$).

Second, stratification of this analysis by current asthma showed that in centers with a high community prevalence of asthma, both asthmatic and nonasthmatic subjects reported more mold exposure in their homes ($r_s = 0.46$ and 0.54, respectively; $P < .01$). This suggests that the observed association between mold exposure and asthma at the individual

level is unlikely to be fully explained by overreporting of mold exposure by asthmatic subjects or underreporting of mold exposure by symptom-free individuals.

Third, the association of reported mold exposure with current asthma at the individual level was evaluated for each study center (Fig 2).

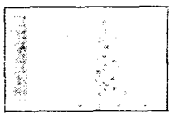

Fig. 2. Reported mold exposure in the last year and current asthma by study center (ranked from low to high according to the frequency of mold exposure). ORs and 95% CIs, adjusted within study centers for sex, age group, and smoking status, are shown. The size of each *square* is proportional to the reciprocal of the variance of the estimate for the center. The *diamond* indicates 95% CI of the combined OR from the model, with study center as the random effect. Thirty-six centers could be included ($P = .56$, test for heterogeneity).

Results for 2 centers had to be excluded in the meta-analysis because of limited numbers of exposed asthmatic subjects. Fig 2 shows a homogeneous positive association between reporting mold exposure and asthma among the centers, being independent of the frequency of reported mold exposure at the center level.

We stratified the analysis between reported mold exposure and asthma according to mold sensitization. Participants who had specific IgE levels to *C herbarum* were at a significantly (P for interaction = .004) increased risk of current asthma if they reported mold exposure (OR, 2.41; 95% CI, 1.32-4.39) compared with those who did not have specific IgE to *C herbarum* (OR, 1.12; 95% CI, 0.97-1.30). Comparable results were obtained after stratifying the analysis between mold exposure and asthma by sensitization according to skin testing to *A alternata*, *C herbarum*, or both. The association of reported mold exposure with asthma was stronger among mold-sensitized subjects (OR, 1.70; 95% CI, 1.14-2.53) than among nonsensitized individuals (OR, 1.17; 95% CI, 1.02-1.36), but the difference was not statistically significant (P for interaction = .33). Findings for wheezing and bronchial responsiveness were less consistent.

Table III shows the housing characteristics associated with reported mold exposure in the last year.

Table III. Housing characteristics associated with reported mold exposure in the last year (N = 16,687)

Housing characteristic	No.	Mold exposure (%)	OR*	(95% CI)
Flat or other building	8347	17.2	1	
House without basement	5834	27.1	1.03	(0.91-1.17)
House with dry basement	2115	22.0	1.24	(1.07-1.44)
House with wet basement	391	44.5	2.10	(1.64-2.68)
House built 1981 or later	3076	13.3	1	
House built 1971-1980	3004	17.9	1.38	(1.19-1.60)
House built 1961-1970	2737	21.7	1.60	(1.38-1.86)
House built before 1960	7870	26.8	1.78	(1.56-2.02)
No water damage in last year	14,702	19.0	1	
Water damage in last year	1985	43.6	3.23	(2.90-3.60)
No central heating	6243	26.8	1	
Central heating	10,444	18.9	0.72	(0.64-0.81)
No ducted air heating	15,460	21.2	1	
Ducted air heating	1227	30.3	0.65	(0.54-0.77)
No extractor fan over cooker	9426	24.5	1	
Extractor fan over cooker†	7261	18.5	0.78	(0.71-0.85)

*ORs for mold prevalence, adjusted for all other listed housing characteristics (each $P < .05$) and study center, are shown.
†Only fans that take the fumes outside the house were taken into account.

The strongest independent associations with mold exposure were type of housing, a home of more than 10 years old, and recent water damage. The presence of central or ducted air heating and of an extractor fan over the cooker seemed to be protective against mold growth. Carpeting was not associated with reported mold exposure.

Discussion

This cross-sectional community-based study among young adults from 18 different countries showed that reporting of mold exposure in homes was associated with current asthma symptoms and bronchial responsiveness. This relationship was consistent across study areas and more pronounced in individuals sensitized to mold. Associations of recent water damage with symptoms were explained by the presence of mold. Asthma symptoms and bronchial responsiveness were less common in individuals with textile floor covers in the bedroom, particularly among those sensitized to house dust mite.

The observed associations between mold exposure and wheezing confirm the results of epidemiologic studies among adults in Finland,[1] [4] the United Kingdom,[2] [3] [7] the Netherlands,[5] and Canada.[6] Some of these studies also found a significant association between mold exposure and asthma, but the present analysis is probably the first to show a relationship between mold exposure and bronchial responsiveness, being homogeneous among countries in different parts of the world. A recent history of water damage was also associated with asthma, but lack of association in multiple models adjusting for mold exposure suggested that this effect was caused by mold growth as a result of home dampness. This was supported by the fact that mold in the home could be predicted by a recent history of water problems in combination with insufficient heating and ventilation, resulting in home dampness.

Relationships between mold exposure and asthma might have different underlying effect mechanisms. First, fungi are sources for allergens that might cause allergic asthma. It is, however, often difficult to relate asthma to mold sensitization or mold exposure because the majority of individuals sensitized to mold are also sensitized to other inhalant allergens.[16] Nevertheless, our study indicated that mold-related effects on asthma were stronger among individuals with specific sensitization to the common molds *A alternata*, *C herbarum*, or both. Both fungal genera have been identified from indoor samples. [17] [18] This suggests that a type I allergic reaction to mold might play a part in the observed relationships between indoor mold exposure and asthma.

Second, the cell wall of most fungi contains $\beta(1\rightarrow3)$-glucans, glucose polymers with inflammatory properties after inhalation.[19] It has recently been shown that glucans in house dust might enhance airway inflammation in asthmatic children.[20] In addition, fungi might also produce mycotoxins, which have been suggested to play a part in nonallergic respiratory symptoms.[16]

Finally, fungi produce a variety of volatile organic compounds[16] that might aggravate a preexisting asthma condition after an irritative mechanism. We speculate that the relationships between indoor mold exposure and current asthma are caused by a combination of allergic and nonallergic mold-related effects, leading to both new-onset asthma and aggravation of asthma. The latter is supported by studies showing that day-to-day severity of asthma was related to variations in airborne fungal spore counts.[21]

Air conditioning was independently associated with an increased prevalence of asthma symptoms but not with bronchial responsiveness. Air conditioning in our study was not related to mold growth, and other studies showed that air conditioning in the home was associated with lower levels of both house dust mites[22] and endotoxins.[23] These indoor contaminants are therefore unlikely to explain the relationship between air conditioning and asthma. We speculate that a changing indoor temperature, humidity, or both as a result of air conditioning might be related to the severity of asthma, but results from other studies are needed to draw conclusions.

We analyzed the relationships between carpeting and asthma. Carpets constitute an important habitat for mites, and carpeting has been shown to be an important predictor of house dust mite allergen levels in homes.[24] In addition, dust from textile floor covers has been shown to contain more microbial contaminants, including fungal propagules[25] and mold components,[26] compared with dust from hard floors. We found a negative association between textile flooring (fitted carpets or rugs) and asthma, being more pronounced for the bedroom than for the living room. The most likely explanation for a negative association would be that symptomatic individuals removed their bedroom carpets in the past. This could be the result of medical advice, especially that given to patients with mite allergy. We speculate that this type of allergen-avoidance measure is more often taken in the bedroom than in the living room because the latter would affect the living comfort of more household members. This is supported by a Dutch case-control study on childhood asthma, in which parents of asthmatic children more often had changed the bedroom floor cover than the living room floor cover for the child's respiratory health. [27] In the Netherlands fitted carpets are common, house dust mite sensitization is prevalent, and allergen-avoidance measures are often advised. An interesting result from our study was that the negative relationship between the presence of bedroom carpets and asthma seemed to be apparent in almost all study centers, including countries in which fitted carpets are uncommon and areas with a low prevalence of house dust mite sensitization. It is therefore unlikely that the observed negative association can be fully explained by a selection effect because of the application of allergen-avoidance measures. Although contradictory with other studies, the cross-sectional design of the present analysis precludes a satisfactory explanation and a statement on a possible causal association.

Exposure to mold was assessed by using questionnaire data, which might have introduced bias because of both differential and nondifferential misclassification. Information on self-reported symptoms and mold exposure was obtained in the same questionnaire. This might have resulted in reporting or recall bias if symptomatic patients overreported the occurrence of mold in their homes, if symptom-free individuals underreported mold exposure, or both. This would have led to biased risk estimates away from the null estimate, which would be consistent with the elevated ORs associated with reported mold exposure. The magnitude of recall bias in this study was reduced by restricting the information on mold levels in homes to the last 12 months and is therefore probably of minor importance. Ecological analyses showed similar relationships between community prevalence of asthma and reported mold exposure for asthmatic and nonasthmatic patients, suggesting that the association observed at the individual level can not be fully explained by overreporting of mold exposure by asthmatic subjects or by underreporting by nonasthmatic subjects. Thus reporting bias might be present, but we do not believe that it fully accounts for the association between mold exposure and asthma.

Misclassification of mold exposure that is not depend-ent on symptomatic status might have led to underestimated risk estimates.[28] Some studies have evaluated the agreement between observed and reported mold exposure and dampness. In a study on adult asthma, reported home dampness was, on average, less than that detected during home visits, both for patients and control subjects. [3] A case-control study in children showed that the parents generally underreported mold growth in the house[29] compared with observations done by trained investigators who were blinded regarding the case-control status. The level of underreporting was not related to the child's respiratory health status. The agreement between observed and reported mold exposure in this study was fair (Cohen's $\kappa = 0.68$), and sensitivity and specificity amounted to 73% and 93%, respectively. However, several investigations have demonstrated that measured levels of fungal propagules in indoor air or in house dust cannot be well predicted by home characteristics[25] [30] [31] These findings suggest that nondifferential misclassification of mold exposure is likely to occur and that ORs in our study might therefore be underestimated. Objective data on domestic mold exposure in our study were not available, and it was therefore not possible to make a quantitative evaluation of exposure misclassification and the resulting bias.

We conclude that mold exposure in homes has an adverse effect on adult asthma symptoms and bronchial responsiveness. This effect was consistent across different countries and was unlikely to be caused by bias. Future research into the mechanisms of mold-related asthma is recommended. There is a need for prospective studies to distinguish between new-onset asthma and aggravation of asthma caused by indoor mold exposure.

We thank Colette Baya and Dr Manuel Hallen for their help during the study and Professor K. Vuylsteek and the members of the COMAC for their support.

References

1. Kilpelainen M, Terho EO, Helenius H, Koskenvuo M. Home dampness, current allergic diseases, and respiratory infections among young adults. Thorax 2001;56:462-7. *Full Text*

2. Evans J, Hyndman S, Stewart-Brown S, Smith D, Petersen S. An epidemiological study of the relative importance of damp housing in relation to adult health. J Epidemiol Community Health 2000;54:677-86. Abstract

3. Williamson IJ, Martin CJ, McGill G, Monie RDH, Fennerty AG. Damp housing and asthma: a case-control study. Thorax 1997;52:229-34. Abstract

4. Pirhonen I, Nevalainen A, Husman T, Pekkanen J. Home dampness, moulds and their influence on respiratory infections and symptoms in adults in Finland. Eur Respir J 1996;9:2618-22. Abstract

5. Brunekreef B. Damp housing and adult respiratory symptoms. Allergy 1992;47:498-502. Abstract

6. Dales RE, Burnett R, Zwanenburg H. Adverse health effects among adults exposed to home dampness and molds. Am Rev Respir Dis 1991;143:505-9. Abstract

7. Platt SD, Martin CJ, Hunt SM, Lewis CW. Damp housing, mould growth, and symptomatic health state. BMJ 1989;298:1673-8. Abstract

8. Burney PGJ, Luczynska C, Chinn S, Jarvis D. The European Community Respiratory Health Survey. Eur Respir J 1994;7:954-60. Abstract

9. Norbäck D, Bjornsson E, Janson C, Palmgren U, Boman G. Current asthma and biochemical signs of inflammation in relation to building dampness in dwellings. Int J Tuberc Lung Dis 1999;3:368-76. Abstract

10. Björnsson E, Norbäck D, Janson C, Widström J, Palmgren U, Ström G, et al. Asthmatic symptoms and indoor levels of micro-organisms and house dust mites. Clin Exp Allergy 1995;25:423-31. Abstract

11. Gehring U, Heinrich J, Jacob B, Richter K, Fahlbusch B, Schlenvoigt G, et al. Respiratory symptoms in relation to indoor exposure to mite and cat allergens and endotoxins. Eur Respir J 2001;18:555-63. Abstract

12. Dharmage S, Bailey M, Raven J, Mitakakis T, Cheng A, Guest D, et al. Current indoor allergen levels of fungi and cats, but not house dust mites, influence allergy and asthma in adults with high dust mite exposure. Am J Respir Crit Care Med 2001;164:65-71. Abstract

13. Janson C, Antó J, Burney P, Chinn S, de Marco R, Heinrich J, et al. The European Community Respiratory Health Survey: what are the main results so far? Eur Respir J 2001;18:598-611. Abstract

14. Roca R, Burgos F, Sunyer J, Saez M, Chinn S, Antó JM, et al. References values for forced spirometry. Eur Respir J 1998;11:1354-62. Abstract

15. Der Simonian R, Laird N. Meta-analysis in clinical trials. Control Clin Trials 1986;7:177-88. Abstract

16. Verhoeff AP, Burge HA. Health risk assessment of fungi in home environments. Ann Allergy Asthma Immunol 1997;78:544-54. Abstract

17. Koch A, Heilemann KJ, Bischof W, Heinrich J, Wichmann HE. Indoor viable mold spores: a comparison between two cities, Erfurt (eastern Germany) and Hamburg (western Germany). Allergy 2000;55:176-80. Abstract

18. Garrett MH, Rayment PR, Hooper MA, Abramson MJ, Hooper BM. Indoor airborne fungal spores, house dampness and associations with environmental factors and respiratory health in children. Clin Exp Allergy 1998;28:459-67. Abstract

19. Rylander R. Investigations of the relationships between disease and airborne (1→3)-β-D-glucan in buildings. Mediators Inflamm 1997;6:275-7.

20. Douwes J, Zuidhof A, Doekes G, van der Zee SC, Wouters I, Boezen MH, et al. (1→3)-β-D-glucan and endotoxin in house dust and peak flow variability in children. Am J Respir Crit Care Med 2000;162:1348-54. Abstract

21. Delfino RJ, Zeiger RS, Seltzer JM, Street DH, Matteucci RM, Anderson PR, et al. The effect of outdoor fungal spore concentrations on daily asthma severity. Environ Health Perspect 1997;105:622-35. Abstract

22. Lintner TJ, Brame KA. The effects of season, climate, and air-conditioning on the prevalence of *Dermatophagoides* mite allergens in household dust. J Allergy Clin Immunol 1993;91:862-7. Abstract

23. Gereda JE, Klinnert MD, Price MR, Leung DY, Liu AH. Metropolitan home living conditions associated with indoor endotoxin levels. J Allergy Clin Immunol 2001;107:790-6.

24. Van Strien RT, Verhoeff AP, Brunekreef B, van Wijnen JH. Mite antigen in house dust: relationship with different housing characteristics in the Netherlands. Clin Exp Allergy 1994;24:843-53. Abstract

25. Verhoeff AP, van Wijnen JH, van Reenen-Hoekstra ES, Samson RA, van Strien RT, Brunekreef B. Fungal propagules in house dust. II. Relation with residential characteristics and respiratory symptoms. Allergy 1994;49:540-7. Abstract

26. Douwes J, van der Sluis B, Doekes G, van Leusden F, Wijnands L, van Strien R, et al. Fungal extracellular polysaccharides in house dust as a marker for exposure to fungi: relations with culturable fungi, reported home dampness, and respiratory symptoms. J Allergy Clin Immunol 1999;103:494-500. Full Text

27. Verhoeff AP, van Strien RT, van Wijnen JH, Brunekreef B. House dust mite allergen (Der p I) and respiratory symptoms in children: a case-control study. Clin Exp Allergy 1994;24:1061-9. Abstract

28. Armstrong BG. The effects of measurement error on relative risk regressions. Am J Epidemiol 1990;132:1176-84. Abstract

29. Verhoeff AP, van Strien RT, van Wijnen JH, Brunekreef B. Damp housing and childhood respiratory symptoms: the role of sensitization to dust mites and molds. Am J Epidemiol 1995;141:103-10. Abstract

30. Ren P, Jankun TM, Belanger K, Bracken MB, Leaderer BP. The relation between fungal propagules in indoor air and home characteristics. Allergy 2001;56:419-24. Abstract

31. Dales RE, Miller D, McMullen E. Indoor air quality and health: validity and determinants of reported home dampness and moulds. Int J Epidemiol 1997;26:120-5. Abstract

Appendix

Principal participants of the ECRHS study

Coordinating Center (London): P. Burney, S. Chinn, C. Luczynska, D. Jarvis, E. Lai; Australia: M. Abramson, J. Kutin (Melbourne); Belgium: P. Vermeire, F. van Bastelaer (Antwerp South, Antwerp Central); Denmark: N. Nielsen (Aarhus); Estonia: R. Jogi (Tartu); France: J. Bousquet (Montpellier), F. Neukirch, R. Liard (Paris), I. Pin, C. Pison (Grenoble), A. Taytard (Bordeaux); Germany: H. Magnussen, D. Nowak (Hamburg); H. E. Wichmann, J. Heinrich (Erfurt); Iceland: T. Gislason, D. Gislason (Reykjavik); India: R. Chowgule (Bombay); Ireland: J. Prichard, S. Allwright, D. MacLeod (Dublin); Italy: M. Bugiani, C. Bucca, C. Romano (Turin), R. de Marco, V. Lo Cascio, C. Campello (Verona), A. Marinoni, I. Cerveri, L. Casali (Pavia); The Netherlands: B. Rijcken, A. Kremer (Groningen, Bergen-op-Zoom, Geleen); New Zealand: J. Crane, S. Lewis (Wellington, Christchurch, Hawkes Bay); Norway: A. Gulsvik, E. Omenaas, C. Svanes (Bergen); Spain: J. Antó, J. Sunyer, J. Soriano, A. Tobías, J. Roca, M. Kogevinas (Barcelona), N. Muniozguren, J. Ramos González, A. Capelastegui (Galdakao), J. Martinez-Moratalla, E. Almar (Albacete), J. Maldonado, A. Pereira, J. Sánchez (Huelva), F. Payo, I. Huerta (Oviedo); Sweden: G. Boman, C. Janson, E. Björnsson (Uppsala), L. Rosenhall, E. Norrman, B. Lundback (Umea), N. Lindholm, P. Plaschke (Goteborg); Switzerland: U. Ackermann-Liebrich, N. Künzli, A. Perruchoud (Basel); United Kingdom: M. Burr, J. Layzqll (Cardiff), R. Hall (Ipswich), B. Harrison (Norwich), J. Stark (Cambridge); United States: S. Buist, W. Vollmer, M. Osborne (Portland).